Network Health Dietitians

Contributor submission form

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PHOSPHATE MANAGEMENT IN CHRONIC KIDNEY DISEASE: IS IT JUST ABOUT THE DAIRY FOODS?

The renal dietitian plays a pivotal role in managing high serum phosphate levels in chronic kidney disease (CKD). Reducing the phosphate content of the diet is essential in treating hyperphosphataemia although this does not come without challenges. By successfully managing phosphate as part of a team, the outcome should be to reduce mortality through preventing incidences of hyperparathyroidism, mineral and bone disorder, vascular calcification and cardiovascular events (1, 2).

Chronic Kidney disease

There is limited prevalence data on CKD for the UK. The NHS Kidney Care report (2012) states that in England approximately 1.8 million people have been diagnosed and are undergoing treatment for CKD. CKD is usually asymptomatic so it is of no surprise when the report states that a further 1 million people may still be undiagnosed(3). Blood pressure management and blood tests have been introduced in screening clinics to try and combat this.

The risk of kidney disease rises rapidly with age and almost half of the patients commencing dialysis are aged 65 years old and over. Many patients requiring dialysis have other co-morbidities such as diabetes and cardiovascular disease, which are also major causes of CKD (4)

CKD is defined as either kidney damage or glomerular filtrate rate (GFR) <60ml/min/1.73m^2 on two consecutive readings for more than 3 months (5). The stages are shown in Table 1.

Phosphate

Phosphate is an essential mineral in the body, mainly found in bones and teeth. Phosphate supports the body to utilise fat, carbohydrates as well as synthesising protein. It is essential for the production of adenosine triphosphate (ATP) which the body uses to store energy. 30% of phosphate in the body is excreted through the intestines and 70% by the kidneys (6). This helps the body to maintain a serum phosphate level between 0.8-1.4mmol/litre.

When the kidneys are healthy parathyroid hormone (PTH), calcitrol; the active metabolite of Vitamin D and Fibroblast Growth Factor 23 (FGF23) maintain calcium and phosphate homeostasis in the body. When serum calcium levels reduce (hypocalcaemia) then PTH is secreted in response, mobilising calcium and phosphate from the bone. Calcitrol is activated in the kidney and increases intestinal absorption of calcium and phosphate. FGF23 is released from the bone in response to elevated serum phosphate levels. FGF23 is sensitive to changes in phosphate and is thought to be an early biomarker of excess phosphate load, although its response is slow and progressive (7).

PTH increases renal reabsorption of calcium while along with FGF23, reduces renal reabsorption of phosphate. Once serum calcium and phosphate levels are corrected PTH and FGF23 are suppressed.

As kidney function declines PTH and FGF23 maintain their role to reduce renal reabsorption of phosphate. Once GFR is less than 20-25ml/min/1.73m^2 the kidney can no longer excrete phosphate resulting in raised serum phosphate levels (8).

Dietary requirements

Evidence suggests that the focus of phosphate management is more important in the early stages of CKD in order to reduce long term complications and delay progression of kidney disease (9, 10). The latest guidelines produced by Kidney Disease Improving Global Outcomes (KDIGO) (11) recommend prescribing dietary phosphate restriction alone or alongside phosphate binders. The guidelines however do acknowledge that the level of evidence is weak and high quality RCTs are required to support this**.**

The renal dietitians role in serum phosphate management is paramount in achieving a positive outcome. The role of the renal dietitian is to advise patients on phosphate containing foods, suitable alternatives and how to match the dose of phosphate binders to the phosphate content of the meal (12).

National and international guidelines recommend a daily dietary allowance of 800mg – 1000mg of phosphate to maintain serum levels of phosphate of 0.9-1.5 mmol/litre for CKD 3b-5 (not on dialysis) and 1.1-1.7mmol/litre for CKD 5D (on dialysis) (11,13,14).

It is also important to consider the fine balance of restricting phosphate intake whilst maintaining sufficient dietary calorie and protein intake to prevent malnutrition. Shinabergar (2008) showed that low serum phosphate levels and low dietary protein intakes in patients with CKD are associated with increased mortality and protein energy wasting. It is important to consider the current recommendations for protein when reducing phosphate content of the diet;

- 0.75 g/kg IBW/day for patients with stage 4-5 CKD not on dialysis (15)
- 1.0-1.2 g/kg IBW/day for patients treated with dialysis (15,16)

Table 2 explains IBW.

Dietary management

Phosphate is present in protein rich foods such as meat, offal, fish, eggs, milk, milk products and pulses. Table 3 shows the phosphate content of most common foods.

In a healthy individual up to 80% of phosphate is absorbed from foods in the intestine (1) which is limited in patients with CKD due to the reduced availability of calcitrol. Most patients with CKD are prescribed a vitamin D

analogue which is usually in the form of 1-alfacalcidol® or zemplar®. This medication improves the patients' ability to absorb up to 60% of phosphate via the intestine. Phosphate content of a grain based source has reduced bioavailability due to the presence of phytate. This can also affect the uptake of calcium and magnesium (10).

As well as considering the natural dietary phosphate from foods, foods that have phosphorus-containing additives also need to be accounted for. Table 4 shows the foods that have phosphorus-containing additives.

Phosphorus-containing additives are especially important in the meat industry where they are used as preservatives (18,19,20). They increase water holding capacity and meat pH, reduce cooking losses, maintain colour, improve protection against microbial growth and improve textural properties (18). The EU has prohibited the addition of phosphates in raw meats but allows it in cooked meats. It is suggested that the phosphate in the foods containing these additives are almost completely absorbed (18,19,21). Research shows that intake from these foods contribute an additional 1000mg/day to a patient's normal intake (18).

High phosphate intakes have been related to a poorer quality diet. Studies conducted in the USA related subjects from a low socio-economic group to have a higher phosphate intake due to the additives in the foods they consumed (1). Indeed with counselling patients were able to lower their phosphate level but it was still a challenge due to the lack of information provided by the food industry. There is also a lack of awareness in some health professionals regarding the addition of phosphates in the form of a preservative (21).

The addition of phosphate as a preservative complicates the patients' ability to minimise phosphate intake. The challenge increases due to the lack of information on food labels about the total phosphorus content of foods. Even food composition tables do not account for the addition of phosphate to foods. Studies have related increased serum phosphate as a cardiovascular risk not only in patients with CKD but also in patients who have normal renal function (20, 22). This supports the need for more public information on the risk of excessive phosphate intake and consideration of comprehensive labelling.

Dialysis

Haemodialysis and peritoneal dialysis is limited in its ability to reduce the total phosphate as a significant amount is found intracellularly. Phosphate tends to be cleared more efficiently in the first half of the treatment compared to the second. Therefore diet and phosphate binder medication are the most effective way of treating hyperphosphataemia.

Phosphate binders

Over the past few years pharmacological treatment in phosphate management has expanded and remains a competitive market. Phosphate

binders are usually introduced when dietary management is limited and the serum levels remain above 1.70mmol/litre. Initial binder therapy includes calcium based phosphate binders such as calcium carbonate (calcichew®) or calcium actetate (phosex®). If the serum phosphate levels remain excessive or serum calcium levels increase then an alternative product can be considered either solely or as a combination with the calcium based binder. Sevelamer carbonate (Renagel®) or lanthanum carbonate (Fosrenol®) are examples of non calcium based binders. Companies have produced medications that can be swallowed, crushed, chewed, or mixed with food or water. While some of these preparations offer patients an alternative to tablets, this still does not reduce the drug burden patients have to persevere with on a daily basis.

Studies have shown that each phosphate binder has a different binding capacity (23). The dietitian will discuss the timing and dose of the phosphate binders based on the patients' meal pattern. Prescribing phosphate binders is still an area of development within the profession.

Adherence

The Renal Registry reported that in the UK 56% of haemodialysis patients and 69% of peritoneal dialysis patients achieved the audit measures for phosphate in 2010 (24). Non- adherence is a common factor in any chronic disease. Many patients with CKD have multiple dietary restrictions, a number of medications to take and possible multiple co-morbidities to cope with (25).

The Health Foundations "Closing the Gap" through changing relationships programme has been introduced to try and enable the patients to take more of an active role in their treatment. By sharing the care the aim is to improve the experience for both patient and staff. It is important that the patient is fully informed about their treatment, including diet and medications, to empower and help them with decision making.

A patient centred approach using good communication skills is vital for a successful outcome. Knowledge is important but unless the patient is engaged and motivated, this alone does not improve a patients' success. Different educational strategies have shown positive outcomes in the short term however for the longer term patients need to be able to self manage (26).

At Leeds Teaching Hospitals renal unit we have started to recognise our patients' achievements. A patient is nominated by a member of the multi-disciplinary team. A poster is then developed so that the nominated patients' success can be shared. The aim is to encourage and motivate other patients on the unit. Figure 1 illustrates one of the stories of success. Consent was gained from the patient for the purpose of reproduction in this article.

Summary

The risks of hyperphosphataemia in CKD are well documented. Diet, medication and dialysis are recognised as essential factors in the control of serum phosphate levels. The challenge in a patient achieving and maintaining acceptable serum phosphate levels however, remain. Empowering the patient to share the responsibility of their treatment needs to central to achieving successful outcomes.

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Tables for article

Table 1 classification of chronic kidney disease (5)

Stage	GFR (ml/min/1.73m^2)	Description
1	>90	Normal or increased GFR, with other evidence of kidney damage
2	60-89	Slight decrease in GFR, with other evidence of kidney damage
3A	45-59	Moderate decrease in GFR, with or without other evidence of kidney damage
3B	30-44	
4	15-29	Severe decrease in GFR, with or without other evidence of kidney damage
5	<15	Established renal failure
5D	<15	Receiving renal replacement therapy

Table 2 Ideal body weight (15)

Ideal body weight should be calculated from height and 'ideal' BMI. 'Ideal' would be:

- 20 for those with actual BMI < 20
- 25 for those with BMI > 25
- actual BMI if it lies between 20-25

Table 3: phosphate content of commonly consumed foods (6)

Food	Phosphate content mmol/l/100g	Portion size	Phosphate content (mmol/l)
Meat			
Lamb/Pork/Beef/Chicken	7	100g	7
Offal	11-16	100g	11-16
Pate	14.5	80g	12
Fish			
Cod/Haddock/Halibut	7	100g	7
Scampi	10	170g	17
Pilchards	9	215g	19
Diary foods and Eggs			
Milk	3	195g	5
Egg	6.4	50g	3.2
Double cream	1.7	15g	0.25
Condensed Milk	8.7	15g	1.3
Hard cheese	16	30g	5
Cream cheese	3.2	30g	1
Cottage cheese	5.2	30g	1.6

Processed cheese/spread	25	30g	7.5
Puddings			
Ice cream	2.9	2 scoops (120g)	3.5
Rice Pudding	2.7	150g	4
Custard	3.5	120g	4.3
Yoghurt	3.1	125g	3.4
Miscellaneous			
Oat based biscuits	7	30g	2.1
Plain sweet biscuit	2.8	20g	0.6
Plain potato crisp	3.5	40g	1.5
Tortilla chips	7.7	50g	4
Peanuts	14	25g	3.5
Sunflower seeds	21	16g	3.3
Ready-Brek (milk)	13.5	180g	24
Porridge (water or milk)	1.5 or 4.6	160g	2 or 7

Table 4 (17, with permission)

Phosphate Additive	Examples of Foods
Diphosphates	Cakes, instant mashed potatoes and cheese
Triphosphates	Fish fingers
Polyphosphates	Dried foods and desserts
Monostarch Phosphates	Ice cream, Pizza, Battered fish, salad dressing
Phosphoric Acid	Beer, processed meats e.g. sausages, sweets, cakes, chocolates, carbonated drinks, jams, vegetable fats and oils
Calcium phosphate	Self-raising flour, cake and pancake mixes, powdered milk drinks, instant pasta and sauces
Ammonium Phosphate	Baked goods, alcoholic beverages, condiments, puddings, baking powder, frozen desserts, margarine, whipped toppings and yeast foods
Magnesium Phosphate	Salt substitute, prepared mustard
Dicalcium Diphosphate	Cupcake mixes, pie tops, instant pasta and sauces, muesli bars, ice cream and instant soups
Phosphate Distarch Phosphate	Batters for frozen foods, custards, sauces, mayonnaise, salad dressing, pies and fillings, instant beverages, dried foods, drinking yoghurt, flavoured milk, whipped cream, coffee, pre cooked pasta and noodles, starch based puddings